majority of instances heal themselves after repair of the hernia. They occur at the columnal squamous junction rather than at the gastro-esophageal junction and in those cases which do not spontaneously heal, a few esophageal dilations are generally sufficient to overcome the narrowing of the esophagus provided the hiatal hernia repair is adequate.

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Lung Oxidant Toxicity

IN CERTAIN EXPERIMENTAL and clinical situations inhalation of oxidants, including both oxygen at tensions considerably above ambient levels and the pollutant oxidants (ozone and oxides of nitrogen) leads to "diffuse bronchio-alveolar damage." Although temporal pathological studies of oxidant-induced lesions in man are meager and the relationship of pulmonary O_2 cytotoxicity to the toxic effects of pollutant oxidants is still unclear, available studies indicate that excessive oxidant exposure causes damage to bronchial mucosa, alveolar epithelium, and capillary endothelium resulting in bronchio-alveolar inflammation and edema. The precise time-dose relationship for the three oxidants is not well defined in man. Experimental and clinical observations suggest a wide range of species and individual susceptibility or resistance to oxidant damage. In addition, tolerance and cross-tolerance to a toxic oxidant level after prolonged low-dose or acute intermittent-dose oxidant exposure has been noted. Although the critical mode of interaction between oxidants and lung cells is still unclear, excessive oxidant exposure can be expected to cause loss of cellular reducing equivalents and unsaturated lipid oxidation, presumably via free radical mechanisms.

Oxidant damage occurs coincident with oxidant interaction with lung cellular oxido-reductive processes and inactivation of enzymatic thiol groups. Recent studies have suggested that the lung contains a reservoir of thiol protective substances (for example, glutathione) and that this reservoir can be compromised or augmented under certain conditions . . . a function of oxidative-protective thiols analogous to those of erythrocytes. Further studies have emphasized the presence in lung tissue of oxidant detoxification mechanisms that potentially function to dissipate the toxic products of lipid peroxidation. Indeed, in tolerance-producing situations these mechanisms show an augmented ability to detoxify lipid peroxides via the glutathione peroxidase mechanism.

Lung enzymatic adaptations may determine lung resistance to oxidant damage . . . increased oxidant levels may induce an augmentation of these lung antioxidant defense mechanisms. Biochemical studies of tissue may be a simple but sensitive indicator for detecting lung oxidant damage. Although current studies are not sufficient by themselves to lead to a unitary version of lung oxidant damage, they indicate a clear program of experiments to test the significance of oxidant induced lung metabolic pertubations and suggest mechanisms of potentially treating these syndromes.

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